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Scott -

Mark

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Mark,

Per our conversations yesterday, I heard back from Dr. Judith Zelikoff (Toxicologist, NYU) who has been one of my key advisors in all of my water contamination work with Water Defense.

Given that we now know that the Particulate Lead is a problem in Flint and it appears that it is not going away, Dr. Zelikoff actually wrote a paper on Inhalation of Particulate Lead some time ago and the abstract is below. Dr. Zelikoff is very concerned that the people in Flint are inhaling Particulate Lead in the showers/baths and they may have been doing so for quite some time. Since there is no air sampling of bathrooms in Flint, this seems to be an unknown. Question: Is it possible for the EPA to meet me this weekend in homes that I have tested and found the volatiles in the showers / baths and set up Air Sampling? Dr. Zelikoff and I believe that air sampling should be set up in the bathrooms/showers in all homes reporting health problems as soon as possible.

As to the volatiles (Chloroform, TTHM's, 1,4 dichlorobenzene etc.) and we have been discussing the Open-Cell Matrix of the WaterBug, it now appears to Dr. Zelikoff and I that the WaterBug may be acting as an air sampler too - mimicking what people are actually breathing into their lungs. In our discussion yesterday, the EPA brought up the comparative data on TTHM's for Flint vs. other cities. I understand this; however, this data appears to be based on sampling from the municipal treatment plants – and not from showers/bath water that is hot and goes through a comprised plumbing system (where chlorine is reacting with organic matter to create the volatile chemicals) as in Flint where people are exposed via

breathing and skin contact. I believe citing TTHM data from other cities may not be relevant for what we are seeing in Flint because of all the variables in Flint and so many things we really don't know but are trying to figure out. Furthermore, the volume of the significant health effects being reported in Flint seem to support that something different is happening in Flint. Note that we do not believe that the WaterBug will detect volatilized Particulate Lead and that is why we need to start doing conventional air sampling in bathrooms in Flint alongside of everything else we are doing.

Also, please note that since we have proven with our independent Lab (ALS Environmental) that the Open-Cell Matrix of the WaterBug retains volatiles and other chemicals after lab extraction, we are understating any concentrations we cite from the lab reports.

Dr. Zelikoff continues to restate everything I have been saying about exposure pathways with breathing/inhalation and skin/dermal and that there are no standards for bathing or showering. This goes to the point I keep reiterating about basing the safety of bathing/showering on drinking water standards – which I continue to believe is comparing apples to oranges.

The abstract of Dr. Zelikoff's paper is below and please let me know if the EPA can meet me at any homes this weekend.

## **Inhalation of particulate lead oxide disrupts pulmonary macrophage-mediated functions important for host defense and tumor surveillance in the lung.**

Zelikoff JT<sup>1</sup>, Parsons E, Schlesinger RB.

### **Author information**

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### **Abstract**

Lead, an immunomodulator and potential human carcinogen, is a major airborne pollutant in industrial environments which poses a serious threat to human health. Despite the wide-spread occurrence of respirable lead particles in the air, and the potential human health risks, effects associated with inhalation of particulate lead on the the lung have been poorly studied. This study was performed to determine whether inhalation of particulate lead oxide (PbO), at a concentration below the currently acceptable air lead standard for occupational exposure, disrupts macrophage (M phi) functions important for maintaining pulmonary immunocompetence. These functions include phagocytosis, production of reactive oxygen intermediates, and the biological activity of tumor necrosis factor-alpha (TNF-alpha). Rabbits exposed to PbO at 30 micrograms/m<sup>3</sup> for 4 days (3 hr/day) were sacrificed and their lungs lavaged immediately, 24 hr, and 72 hr after the final exposure. Lactate dehydrogenase (a marker of lung cell damage) and lysozyme activity (a marker of lysosome permeability), measured in the lavage fluid, were significantly increased 24 and 72 hr after exposure. PbO produced neutrophil infiltration nor effects on M phi viability or total numbers. Effects on M phi functions were as follows. Phagocytic uptake of latex particles was reduced with increasing post-exposure time reaching a maximum inhibition at 72 hr. Inhalation of PbO enhanced hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) and superoxide anion radical (O<sub>2</sub><sup>-</sup>) production in a time-dependent manner; effects on H<sub>2</sub>O<sub>2</sub> began at 24 hr and were persistent up to 72 hr. Effects on TNF-alpha release/activity appeared earliest and were persistent up to 72 hr. Immediately and 24 hr after exposure, lipopolysaccharide-stimulated activity of TNF-alpha was depressed by 62 and 50%, respectively; after 72 hr, TNF-alpha release was significantly enhanced compared to control levels. Results demonstrate that the lung is a sensitive target for the toxic effects of inhaled lead. This study provides the first evidence

that inhalation of particulate lead, at an occupationally relevant concentration, and in the absence of elevated blood lead levels, alters pulmonary M phi functions critical for lung defense against inhaled antigens. Our findings may have important implications for human health and should be considered when evaluating the health risks associated with inhaled lead.

**Best Regards,**

**Scott Smith**

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